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CASE REPORT

Rebleed in traumatic subarachnoid haemorrhage

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Introduction

Trauma is the most common cause of subarachnoid haemorrhage.[10,18] This is thought to be caused by a haemorrhagic contusion bleeding into the subarachnoid space (as contusions are the most common accompanying pathology detected on computerized tomography (CT) of the head[5]), or secondary to intraventricular bleeding due to tearing of the tela choroidea.[3] Other rarer causes of traumatic subarachnoid haemorrhage (TSAH) include rupture of the posterior inferior cerebellar artery,[1] intracranial[14] or extracranial vertebral artery,[2] internal carotid artery,[16] carotid-cavernous fistula[13] and traumatic aneurysms[4,15] in penetrating head injuries. TSAH is usually mild and almost never life threatening with low incidence of complications and low mortality.

We describe a unique case of TSAH with normal cerebral angiogram that presented with a further bleed. This complication has not been described before.

Clinical history

A 38-year-old housewife was horse riding with her husband. She was wearing a helmet. The horse suddenly reared causing her to fall off backwards and struck the back of her head. She lost consciousness for about 10 min, and remained very confused and agitated when she regained consciousness. There was no history of urinary incontinence, tongue biting or seizure. There was also no history of a sudden headache prior to the fall.

At the accident scene, her Glasgow Coma Scale (GCS) was 13/15. She was transferred urgently to the nearest Accident and Emergency department (A&E). On arrival, her GCS dropped to 7/15 and she was immediately intubated and ventilated. Examination revealed bruising in her left chest wall with no other injuries. Chest, cervical and pelvic radiographs were done which showed contusions in her left lung, and no fractures.

A computerized tomography (CT) scan of her head was done (Fig. 1), which revealed diffuse subarachnoid blood with blood in both Sylvian fissures, the third and fourth ventricle. There was early dilatation of her temporal horns suggesting early hydrocephalus. There were no signs of any contusions. The appearances were atypical for TSAH, and the suspicion arose that there could have been an aneurysmal SAH.

She was started immediately on nimodipine and then sent to the regional neurosurgical unit. Urgent digital subtraction cerebral angiography (DSA) demonstrated all four vessels with no vasospasm. There was no evidence of an aneurysm or arteriovenous malformation (AVM). The cerebral angiogram was of good quality.

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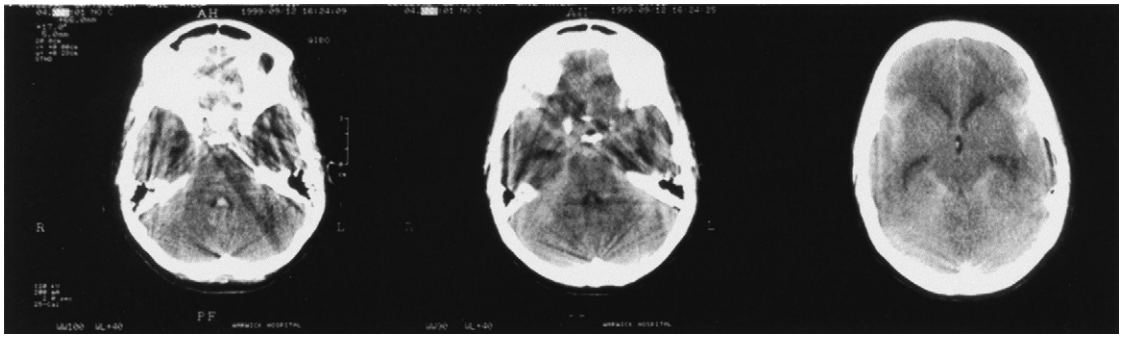

Figure 1 Computerized tomography (CT) scan of the patient's head which revealed diffuse subarachnoid blood in both Sylvian fissures and the fourth ventricle with early dilatation of her temporal horns suggesting early hydrocephalus.

Because of the benign nature of the angiogram, she was woken up and extubated. She was fully alert and orientated with no neurological deficits. Her hydrocephalus caused headaches, treated successfully with serial lumbar punctures. After 10 days in hospital she was well enough to be discharged home. Magnetic resonance imaging with angiography (MRA) done 2 days prior to discharge was normal. There was no evidence of any arterial-venous malformation (AVM), aneurysms or contusions.

Twelve days after her first bleed, she suffered a grand mal seizure during sleep. Her GCS dropped to 3/15 and she was admitted urgently to hospital where she was immediately intubated and ventilated. Repeat CT head showed diffuse subarachnoid haemorrhage with blood in both Sylvian fissures and also in the third, fourth and lateral ventricles with more marked hydrocephalus (Fig. 2). The quantity of blood on the scan indicated that she had had a further bleed. Repeat digital subtraction cerebral angiography again showed no evidence of an aneurysm or AVM.

Twenty-four hours later, she was extubated. She regained full consciousness, and complained of severe headaches. Examination this time revealed decrease visual acuity in her left eye due to a subhyloid haemorrhage. There were no other neurological deficits. Blood investigations revealed normal platelet count and clotting studies.

Because of the hydrocephalus on the scan and her severe headaches, an external ventricular drain was inserted. The opening pressure was well above 30 cm of water. She remained shunt dependent and after 5 days, a lumbo-peritoneal shunt was inserted after repeat CT showed resolution of her interventricular blood.

She made an uncomplicated recovery and was discharged 7 days later. Four months later, she complained of low-pressure postural headache brought on by activity and relieved by resting. The lumbo-peritoneal shunt was removed and her headaches resolved immediately. Magnetic resonance angiography was repeated and remained normal. At follow-up 18 months later, she remained well with no further rebleeds.

Discussion

TSAH is a frequent occurrence in moderate to severe head injuries. In general, the more severe the head injury, the more likely there will be associated TSAH

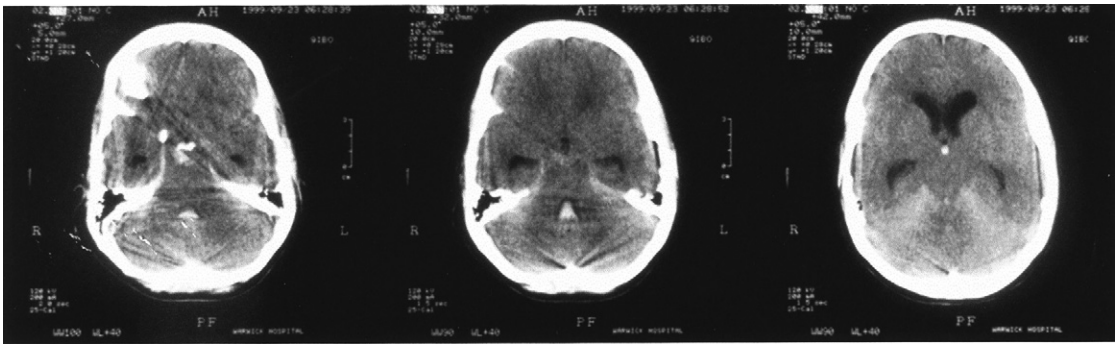

Figure 2 Repeat CT head showed diffuse subarachnoid hemorrhage in both Sylvian fissures and also in the third, fourth and lateral ventricles with more marked hydrocephalus. The quantity of blood on the scan indicated that she had had a further bleed.

and the more the amount of blood seen on CT.[8] Therefore, it is a negative prognostic factor in head injuries[8,12] especially if there is an associated intracerebral haematoma.[5] Although traumatic subarachnoid blood can be found anywhere in the brain, it is usually found in the ambient cisterns, quadrigeminal cistern and/or Sylvian fissure. This distribution of blood is associated with good outcome.[11] Patients, who have subarachnoid blood in the prepontine and/or interpeduncular cisterns, in contrast have markedly poor outcomes,[19] as do those with large amounts of blood on CT.[10] This is presumably related to the force and severity of the head injury.

Compared to aneurysmal subarachnoid haemorrhage (ASAH), TSAH has much lower morbidity and mortality. The complications seen in TSAH are vasospasm causing delayed neurological deficit and communicating hydrocephalus.[5] The vasospasm seen in TSAH is milder; the incidence is lower and less likely to cause cerebral infarction than vasospasm in ASAH.[6] It is also more likely to be localized to one area, particularly in the vertebro-basilar territory.[17] It thought to be caused by the subarachnoid blood irritating the blood vessel, and its severity is proportional to the blood load on CT.[18] As subarachnoid blood disappears more rapidly in TSAH than for ASAH, in most cases the vasospasm is usually short-lived and does not require treatment.[6] The other known complication is communicating hydrocephalus. This is also usually mild and short-lived, requiring no treatment[7] or a series of lumbar punctures. The treatment of TSAH is supportive as for routine head injury patients with particular care to avoid hypotension and hypoxia, which are commoner in this group of patients especially in the acute stage.[9]

We describe a unique case of TSAH in a patient with moderate head injury, which presented with a clinically diagnosed and radiologically confirmed rebleed 12 days after the primary event. Aneurysms, AVMs and arterial dissection had been ruled out with two good quality DS cerebral angiography and two MRA. This has not been encountered before in our clinical practice and never been described before in literature. The cause of her TSAH was speculated to be the rupture or dissection of small cortical vessels due to the shearing stress of her head injury.

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